Effect of a Screening Program on Changing Patterns of Lead Poisoning

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A biphase program of screening and treating high-risk children for lead poisoning resulted in a 30% fall in mean lead values in the target areas over a 5-year period. The mean and median for subjects under 6 years was 4–10 μg/100 ml higher than for those over 6. Median for a high incidence area was 42 μg/100 ml in 1967 and 30.0 in 1971; for a low incidence area, 33 and 20 μg/100 ml in the equivalent years.

Ingestion of lead paint was observed or demonstrated by x-ray in 90% of 2200 patients treated in the Lead Clinic. Gross neurologic sequelae were limited to two cases of mild, persistent ataxia. Impaired intellectual performance was observed subsequently in several asymptomatic patients with initial blood lead levels (PbB) ≥ 100 μg/100 ml.

A concerted effort in Chicago to screen children at risk for lead poisoning and to treat those with elevated blood lead resulted in marked diminution of incidence over a 5-year period in which over 200,000 were tested. All subjects with elevated PbB (blood lead) were referred to the Lead Clinic for evaluation, where treatment was given on an ambulatory basis unless hospitalization was indicated (*1*).

The extent of elevated PbB, as revealed by the screening program, was far greater than anticipated (Table 1). In 1967, the initial year, PbB of 50 μg/100 ml or more was present in 8.5% of children tested; in 1968 the number was reduced to 3.8%, and remained at 2% in each of the subsequent 3 years. In all there were 6800 PbB results over 49 μg/100 ml and three to four times as many between 40 and 49 μg/100 ml.

Of the 6800 subjects with PbB of at least 50 μg/100 ml, there were 77.5% in the 50–59 μg range (Table 2), at least two-thirds of whom came to the Lead Clinic. Of the remaining 22.5%, whose lead values were from 60 to 275 μg/100 ml, clinic attendance was well over 90%. Close to 10,000 children were admitted to the Lead Clinic between January 1967 and December 1971 of whom 1500 had values over 59 μg/100 ml and 8500 from 40 to 59 μg/100 ml.

A history of ingestion of peeling paint and broken plaster was obtained in 80%

Table 1. Number of blood lead specimens ≥50 μg/100 ml detected in screening program, 1967–1971.

Year	Number Screened	Children with PbB ≥50 μg/100 ml	
		number	%
1967	27,959	2,379	8.5
1968	40,785	1,556	3.8
1969	47,527	716	1.5
1970	44,347	911	2.1
1971	52,990	1,242	2.3
Total	213,598	6,804	3.2

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Table 2. Breakdown of 6804 children with blood lead concentration ≥50 μg/100 ml.

PbB, μg/100 ml	Number of children	Per cent of total
50–59	5284	77.5
60–69	666	10.0
70–79	389	5.7
80–89	203	3.0
90–99	87	1.3
100–275	175	2.5
Total	6804	100.0

of the patients. In another 10%, the source of lead was established by disclosure of paint particles in the abdomen on x-ray. Occasionally mothers denied pica for paint and plaster because of embarrassment or guilt. Several mothers confessed to giving the child an enema before coming to the clinic in hope the x-ray would prove negative. An x-ray of the abdomen was taken only once unless elevated blood lead recurred. If it had been possible to repeat x-rays of the abdomen at each clinic visit, evidence of paint ingestion might have been observed in all patients.

Although the screening was directed at a well-child population, 465 of the patients had PbB levels between 80 and 275 μg/100 ml.

This incidence, if the disease had been polio, would have been designated an epidemic and a national emergency would have been declared; however, since lead poisoning is a disease of the poor with deep roots in the politically delicate area of real estate, no alarm was sounded. Housing has never been the subject of a conference such as this.

Dwellings in the target areas date from the late 1800's to 1930. Interior walls are generally of plaster, now crumbling, to which lead base paint had been applied. In other instances, wallpaper was used to cover the paint, or paint was applied over the wallpaper as well as on woodwork. Chicago homes have back porches and back stairs painted, traditionally, battleship gray or dark green. The lead concentration of paint flakes from these surfaces may be over 30%.

This review of the effectiveness of the combined program is limited to the peak months of the summer, when lead poisoning is at its height in number of reported cases, of dangerously high PbB levels, and of patients with symptomatic disease. Three areas in diverse sections of the city were selected for comparison. The first two, with a largely black population, were known as high incidence areas in both mortality and morbidity for some years. The third is a lower incidence area primarily Spanish in composition. Children who had reached their sixth birthday were studied separately because pica is uncommon in the school child and PbB tends to be lower.

Since mean values were so high in the summer of 1967, the results of the following summer were also studied to determine what impact the previous year's effort would have on the prevalence of the disease. Both were then compared to 1971, the fifth year of the combined program.

Area E is one of the more notorious districts (Table 3). In the first summer, PbB values ranged from a low of 12 μg/100 ml, which is less than the mean obtained for rural residents, to a high of 150 μg/100 ml (*2*). Mean and median values were 43.9 and 42 μg/100 ml, respectively, with 55% of test results 40 μg/100 ml and over. By the following summer, the mean was lower by 12 μg but in 1971 it was still 30.7 μg/100 ml. However, the percentage of children showing PbB values over 40μg/100 ml continued to fall, eventually reaching 16%, indicating that even in peak summer the mode had shifted by about 10 μg/100 ml in Area E.

In the older children, the initial range is from 13 μg/100 ml, a low identified with children not exposed to lead, to a high of 63 μg/100 ml. The same rate of diminishing lead values appears here as in the younger children. Presumably the child who was 6 years old in 1971 was detected with incipient lead poisoning at age 3 and prevented from entering the morbid statistics of subsequent years.

Area L, another high incidence district (Table 4) which has suffered increasing economic distress in the past 25 years, reveals a similar trend. Over the 5-year period,

Table 3. Urban lead poisoning in peak summer months; high incidence area E; ethnic predominance black.

Year	Number tested	Range, μg/100 ml	Mean, μg/100 ml	Median, μg/100 ml	% of total with PbB ≥40 μg/100 ml
	<6 Years				
1967	95	12–150	43.9	42.0	55.0
1968	210	10– 78	31.3	32.0	20.0
1971	250	10–160	30.7	30.0	16.0
	>6 Years				
1967	30	13– 59	33.3	34.5	20.0
1968	103	12– 59	25.9	26.0	9.6
1971	58	10– 47	24.0	24.0	8.6

the mean in summer fell from 40.7 to 27.9 μg/100 ml. In 1971, only 10% of young children at risk have PbB levels above 40 μg/100 ml, in contrast to 42.0% 5 years previously. Those over 6 years show a 9 μg/100 ml drop in the mean, and in this group, none appeared in the 40's.

Lead poisoning is not as great a problem in Area D (Table 5), where family relationships are a little more stable and fewer mothers are employed. The mean initially is 33.7 μg/100 ml but after 5 years is down to 23.3 μg/100 ml, exhibiting roughly the same decrease of 30% as the other two districts. Similar improvement occurred in children over 6, whose final mean of 20.8

Table 4. Urban lead poisoning in peak summer months; high incidence area L; ethnic predominance black.

Year	Number tested	Range, μg/100 ml	Mean, μg/100 ml	Median, μg/100 ml	% of total with PbB ≥40 μg/100 ml
	<6 Years				
1967	100	19–200	40.7	36.0	42.0
1968	100	10– 67	35.8	34.0	29.0
1971	100	14– 80	27.9	28.0	10.0
	>6 Years				
1967	30	22– 53	34.2	31.5	13.3
1968	30	13– 41	28.1	28.5	10.0
1971	30	14– 38	24.8	24.0	0

Table 5. Urban lead poisoning in peak summer months; moderately high incidence area D; ethnic predominance Spanish.

Year	Number tested	Range, μg/100 ml	Mean, μg/100 ml	Median, μg/100 ml	% of total with PbB ≥40 μg/100 ml
	<6 Years				
1967	100	12–77	33.7	33.0	21.0
1968	105	10–69	27.2	25.0	12.0
1971	283	10–87	23.3	20.0	5.3
	>6 Years				
1967	30	17–48	25.0	27.0	5.0
1968	102	10–65	25.0	23.0	5.8
1971	56	10–46	20.8	18.0	5.3

μg/100 ml compared very favorably with the mean of 23.5 μg/100 ml obtained in 746 subjects, 10 to 14 years, who resided in the target districts in Chicago in 1967. As an incidental note, this group exhibited the same seasonal variation encountered in children under 6, varying from 19 μg/100 ml in the winter to 26.5 μg/100 ml in summer (*3*).

Symptomatic lead poisoning (Table 6) was discovered by history or by physical examination in 180 of the 2200 patients treated in the clinic in the 5 years from 1967 to 1971. Symptoms attributable to lead toxicity were present in 71 or 12% of all patients treated in 1967, in 32 or 5.6% in 1968, and in 14 or 4.2% in 1971. The total number of patients as well as the number with signs of impending encephalopathy is considerably lower by 1971.

Symptoms (Table 7) are rarely present under PbB levels of 70 μg/100 ml. When they are elicited by history in association with PbB levels of 50 or 60 μg/100 ml, they may well represent concomitant disease. In the presence of other laboratory tests positive for lead poisoning, it may be impossible to differentiate these nonspecific complaints from those common to pediatric practice. In contrast to the few patients with symptoms at low lead levels are the many without evidence of clinical illness despite values as high as 250 μg/100 ml. Symptoms were absent or sufficiently mild in three-fourths of the 177 children with PbB over 100 μg/100 ml to permit them to be treated on an out-patient basis.

Maturational delay in speech and impaired intellectual performance were manifest in several with blood lead levels in excess of 100 μg/100 ml, even where symptoms were never apparent. Psychological testing of several school children who had subclinical lead poisoning at one to three years indicated impaired abstract reasoning and verbalization of concepts.

Mortality figures for Chicago in the preceding 5 years showed 74 recorded deaths. Between 1967 and 1971 there were 35, of which 25 were reported in the first 2 years and 10 in the next 3 years. Only one death occurred in the 200,000 children tested.

Table 6. Incidence of symptoms in first, second, and fifth year of lead poisoning detection program.

Year	No. of patients	No. with symptoms	% with symptoms
1967	582	71	12.2
1968	573	32	5.6
1971	333	14	4.2

Table 7. Symptoms of lead poisoning discovered in a well-child screening program (180 of 2200 patients).

Symptoms in order of frequency	PbB range, μg/100 ml
1. Drowsiness	63–232
2. Vomiting	60–208
3. Irritability	55–192
4. Gastrointestinal (stomach ache, constipation, diarrhea)	67–152
5. Behavior changes (withdrawal, regression, retarded development)	88–208
6. Ataxia	61–208
7. Convulsions	125–175
8. Stupor	119–208
9. Peripheral neuropathy	140

Conclusion

A unified program for detection and treatment of children with elevated PbB resulted in a 30% decrease in mean PbB in the target areas over a 5-year period. Ingested lead-bearing paint was the sole etiologic agent in 90% of the patients as determined by history or by x-ray of the abdomen.

Screening covered only one-fourth of the children at risk yearly. The child who is not detected through screening, and whose pica does not cease spontaneously, is eventually brought to the emergency room with acute encephalopathy. Should pica stop before symptoms appear, PbB will in time recede to normal without lead posioning having been detected. However, when he fails at school several years later, it is erroneously concluded that brain damage may result from low lead level toxicity.

Twenty years ago, Bradley found a mean

PbB of 43 μg/100 ml in 333 children at risk in Baltimore, with values ≥50 μg/100 ml in 44.4% (*4*). A recent study by Fine (*5*) of 6150 children in 14 smaller Illinois cities revealed a 19% incidence of PbB ≥40 μg/100 ml with 4% having PbB ≥60 ≥ μg/100 ml. At the Pediatric Academy sessions in 1972 on "Poison in the Walls" an HEW representative stated there are still 30 to 40 million houses in the country with hazardous lead paint.

Until the overwhelming problem of lead-paint in housing is solved, the contribution of lead to body stores from any other source cannot be correctly gauged. Until then, low level lead toxicity must be considered the endpoint of high level lead toxicity that was not recognized in time.

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